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CASE REPORT

Esophageal tuberculosis; A rare cause of odynophagia: A case report

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Abstract We present a rare case of esophageal tuberculosis causing odynophagia. Tuberculosis of the esophagus is a rare condition, even in countries with a high incidence of tuberculosis (TB) and studies estimate that it constitutes about 0.3% of gastrointestinal TB cases. The case emphasizes the diverse ways tuberculosis can present.

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Our patient was a 24 year old male who presented with 1 year history of upper abdominal discomfort, odynophagia, and post prandial distention. For two months he was also having dry cough, dyspnea on exertion, progressive distention of abdomen and low grade evening rise of temperature. On examination he was found to have grade 1 clubbing, ascites and small axillary lymph nodes.

Hemogram and serum chemistry were normal. HIV serology was also negative.

Chest X ray showed a right hilar lymph node (Fig. 1). USG abdomen confirmed moderate ascites and showed multiple internal septations.

A diagnostic paracentesis showed a lymphocytic exudative ascites with high protein, high lactate dehydrogenase, high adenosine deaminase, and a serum ascites albumin gradient of 0.3. No acid fast bacilli could be demonstrated in the ascitic fluid microscopically but polymerase chain reaction for *Mycobacterium tuberculosis* was positive. There were no malignant cells. Mantoux skin test was negative.

Esophageogastroduodenoscopy showed longitudinal esophageal friable ulcers at 32 cms from incisors (Fig. 2). Biopsy was done which showed chronic granulomatous inflammation (Fig. 3). PCR was positive for *M. tuberculosis* in the esophageal biopsy.

Contrast enhanced computed tomography (CECT) chest showed mediastinal lymphadenopathy with normal lung parenchyma (Fig. 4). CECT abdomen showed peritoneal thickening and ascites with septations.

With these investigations a diagnosis of secondary esophageal tuberculosis with extension from nearby mediastinal

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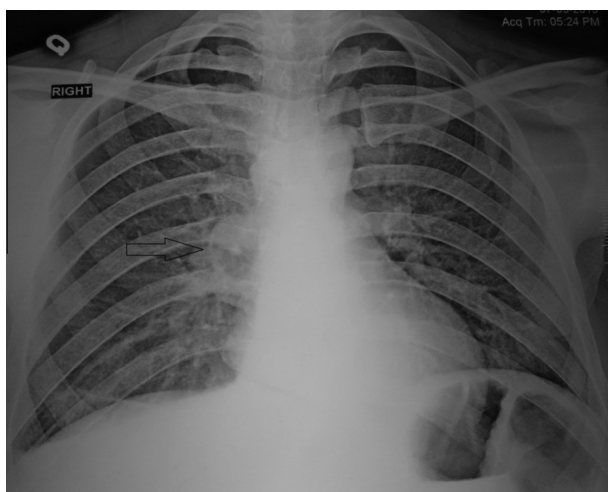


Fig. 1 A chest X-ray showing a right hilar node (arrow).

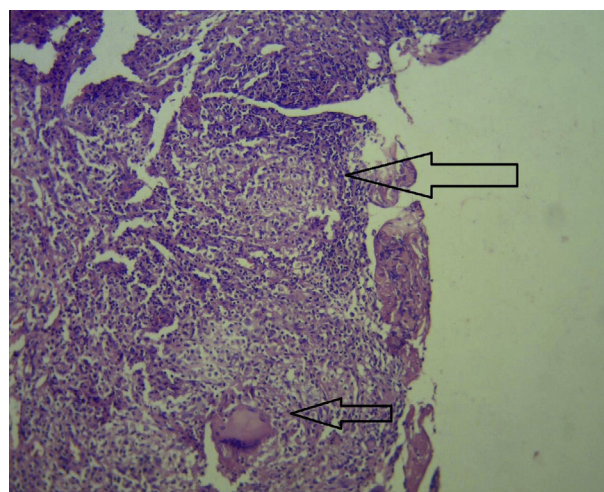


Fig. 3 Esophageal biopsy showing chronic granulomatous inflammation with the top arrow showing a granuloma; and the bottom arrow showing a giant cell.



Fig. 2 Arrow showing the longitudinal esophageal ulcers.

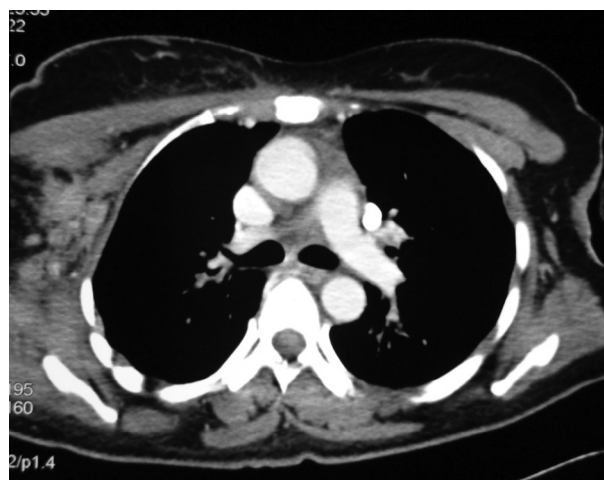


Fig. 4 CECT showing mediastinal lymphadenopathy.

nodes; with tubercular ascites was made. The patient was started on anti tubercular drugs for a total duration of 6 months. At follow up on 2 months the odynophagia and ascites had disappeared; the patient was afebrile and asymptomatic. After 6 months of treatment repeat esophagoscopy was normal with no complications.

Discussion

The global burden of tuberculosis remains enormous, mainly because of poor control and coexisting nature of *M. tuberculosis* and HIV. Tuberculosis has been known to involve the esophagus, either as a primary infection or as a secondary manifestation of reactivated disease. The exposure of the esophagus to the organism is limited by the rapid clearance of infected sputum by means of coordinated peristalsis, combined with upright posture and an intact lower esophageal sphincter [1]. Esophageal tuberculosis is almost always associated with medi-

astinal lymphadenopathy with or without a tracheo-esophageal fistula [2]. The two most common differential diagnoses are carcinoma of the esophagus and Crohn's disease of the esophagus [3,4]. Tuberculosis of the esophagus is a rare condition, even in countries with a high incidence of tuberculosis (TB) [5,6], and studies estimate that it constitutes about 0.3% of gastrointestinal TB cases [7]. Involvement of the gastrointestinal tract occurs through ingestion of infected sputum or hematogenous spread from primary pulmonary TB [8]. Most cases of esophageal tuberculosis are secondary to direct extension from adjacent structures, such as mediastinal lymph nodes or pulmonary sites. Primary esophageal tuberculosis is even rarer [9]. Esophageal involvement by tuberculosis usually affects the middle third of the esophagus at the carina level [10].

The most frequent symptom reported in esophageal tuberculosis is dysphagia, which occurs in about 90% of cases [11]. Other symptoms are odynophagia and retrosternal pain and the occurrence of symptoms such as fever, weight loss and an-

orexia is also common [11]. Choking on swallowing may be indicative of an underlying fistula between the esophagus and respiratory tract. In our patient the predominant symptom was odynophagia. Infection with human immunodeficiency virus, immunosuppressive therapy, and immigration from tuberculosis-endemic areas are risk factors for esophageal tuberculosis [2]. Complications include bleeding, perforation, fistula formation [12], aspiration pneumonia, fatal hematemesis, traction diverticula, and esophageal strictures [13] which were not present in our patient.

Approximately 65% of patients with esophageal tuberculosis have nonspecific findings on chest radiograph suggestive of tuberculosis. Enlarged lymph nodes with a hypodense center on computed tomography scan of the chest that are characteristic of tuberculous lymphadenitis are observed in the majority of these patients [13] as was seen in our patient.

Diagnosis of esophageal tuberculosis can be made by esophagoscopy or biopsy of associated mediastinal lymphadenopathy [11]. A differential diagnosis of esophageal tuberculosis includes esophageal carcinoma, Crohn's disease, moniliasis, actinomycosis, syphilis, and esophageal injury secondary to ingestion of caustic material [13].

Esophageal TB is managed with anti-tuberculous drugs; surgery being reserved for complications including a non-healing tracheoesophageal or bronchoesophageal fistula, stricture, or bleeding from an aorto-esophageal fistula [2,6,11]. A 6-to-9-month course of anti-tuberculous chemotherapy is sufficient for immunocompetent patients treated with a regimen consisting of four first-line drugs, namely isoniazid, rifampicin, ethambutol, and pyrazinamide for the initial 2 months, then continuing with isoniazid and rifampicin for another 4-7 months [2,6,14]. Our patient responded to a 6 month treatment only. It is necessary to give therapy for longer if one or more of these drugs cannot be used because of intolerance or drug resistance [14]. In cases of MDR-TB, defined as TB caused by organisms showing in vitro resistance to at least both isoniazid and rifampicin, the total treatment duration should be extended to at least 18 months, and the regimen should comprise 5 to 6 drugs to which the organisms are susceptible for the initial 6 months, followed by 3 to 4 drugs subsequently [15].

Conflict of interest

None declared.

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